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=> s airway hyperresponsiveness
L1 5682 AIRWAY HYPERRESPONSIVENESS

=> s l1 and inhibitor
L2 1246 L1 AND INHIBITOR

=> s l2 and calcitonin gene related peptide receptor
L3 1 L2 AND CALCITONIN GENE RELATED PEPTIDE RECEPTOR

=> d l3 ibib abs

L3 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
2001:693112 Document No. 135:236441 Role of calcitonin gene-related peptide (CGRP) for reducing allergen-induced **airway hyperresponsiveness**. Gelfand, Erwin W.; Dakhama, Azzeddine (National Jewish Medical and Research Center, USA). PCT Int. Appl. WO 2001068118 A1 20010920, 66 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US8264 20010314. PRIORITY: US 2000-PV189622 20000314.

AB Disclosed is a method to reduce **airway hyperresponsiveness**, such as allergen-induced **airway hyperresponsiveness**, in a mammal by administering an agent that increases the biol. activity of a CGRP receptor. The agent administered to a mammal is an antibody in conjunction with another agents selected from the group consisting of corticosteroids, (oral, inhaled and injected), .beta.-agonists (long and short acting), leukotriene modifiers (**inhibitors** or receptor antagonists), antihistamines, phosphodiesterase **inhibitors**, sodium cromoglycate, nedocromil, theophylline and CGRP. Also disclosed are methods for identifying compds. useful in the present method.

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=> d his

     (FILE 'HOME' ENTERED AT 12:56:10 ON 20 JUL 2002)

     FILE 'MEDLINE, EMBASE, BIOSIS, SCISEARCH, CAPLUS' ENTERED AT 12:56:24 ON
     20 JUL 2002
L1          8882 S AIRWAY HYPERRESPONSIVENESS
L2          1246 S L1 AND INHIBITOR
L3             1 S L2 AND CALCITONIN GENE RELATED PEPTIDE RECEPTOR

=> s l2 and "CGRP" antagonist
L4             0 L2 AND "CGRP" ANTAGONIST

=> s l2 and "CGRP receptor"
L5             1 L2 AND "CGRP RECEPTOR"

=> d l5 ckib abs

L5   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2002 ACS
2001:683112   Document No. 135:236441  Role of calcitonin gene-related peptide
     (CGRP) for reducing allergen-induced airway
     hyperresponsiveness.  Gelfand, Erwin W.; Dakhama, Azzeddine
     (National Jewish Medical and Research Center, USA).  PCT Int. Appl. WO
     2001068119 A1 20010920, 69 pp.  DESIGNATED STATES: W:  AE, AG, AL, AM, AT,
     AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ,
     EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP,
     KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO,
     NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG,
     US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW:  AT, BE, BF,
     BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU,
     MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR.  (English).  CODEN: PIXXD2.
     APPLICATION: WO 2001-US6274 20010314.  PRIORITY: US 2000-PV189622
     20000314.
AB   Disclosed is a method to reduce airway
     hyperresponsiveness, such as allergen-induced airway
     hyperresponsiveness, in a mammal by administering an agent that
     increases the biol. activity of a CGRP receptor.  The
     agent administered to a mammal is an antibody in conjunction with another
     agents selected from the group consisting of corticosteroids, (oral,
     inhaled and injected), .beta.-agonists (long and short acting),
     leukotriene modifiers (inhibitors or receptor antagonists),
     antihistamines, phosphodiesterase inhibitors, sodium
     cromoglycate, nedocrimal, theophylline and CGRP.  Also disclosed are
     methods for identifying compds. useful in the present method.


=> s (Gelfand e?/au or Dakhama a?/au)
L6          2916 (GELFAND E?/AU OR DAKHAMA A?/AU)

=> s l6 and airway hyperresponsiveness
L7           265 L6 AND AIRWAY HYPERRESPONSIVENESS

=> dup remove l7
PROCESSING COMPLETED FOR L7
L8            99 DUP REMOVE L7 (166 DUPLICATES REMOVED)

=> s l8 and calcitonin gene related peptide
L9             2 L8 AND CALCITONIN GENE RELATED PEPTIDE

=> dup remove l9
PROCESSING COMPLETED FOR L9
L10            2 DUP REMOVE L9 (0 DUPLICATES REMOVED)
```

=> d l10 1-2 chib abs

L10 ANSWER 1 OF 2 MEDLINE
2002219008 Document Number: 21952265. PubMed ID: 11956058. Regulation of **airway hyperresponsiveness** by **calcitonin gene-related peptide** in allergen sensitized and challenged mice. **Dakhama Azzeddine**; Kanehiro Arihiko; Makela Mika J; Loader Joan E; Larsen Gary L; **Gelfand Erwin W.** (Division of Cell Biology and Pulmonary Medicine, Department of Pediatrics, National Jewish Medical and Research Center, Denver, Colorado 80206, USA.) AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE, (2002 Apr 15) 165 (8) 1137-44. Journal code: 9421642. ISSN: 1073-449X. Pub. country: United States. Language: English.

AB Sensory neuropeptides are localized to airway nerves and endocrine cells in both human and animal species and may participate in the development of airway inflammation and hyperresponsiveness (AHR). We used a mouse model to identify the changes that occur in **calcitonin gene-related peptide** (CGRP) expression in the airways during development of allergic inflammation and to investigate the potential role of this neuropeptide in modulating AHR. In sensitized mice, allergen challenge induced eosinophilic airway inflammation and AHR and resulted in considerable depletion of CGRP in neuroepithelial bodies and submucosal nerve plexuses without altering the overall density of airway nerve fibers. This depletion was subsequent to the development of airway inflammation and was prevented by anti-very late antigen-4 and anti-interleukin-5 treatments, which blocked airway eosinophilia and abolished AHR. Administration of CGRP to sensitized and challenged mice resulted in the normalization of airway responsiveness to inhaled methacholine, an effect that was neutralized by the receptor antagonist CGRP(8-37). These data demonstrate that replacement of CGRP following its depletion in allergic mice can reverse the changes in airway responsiveness and suggest that CGRP may have potential for the treatment of allergic AHR.

L10 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2002 ACS
2001:693112 Document No. 135:230441 Role of **calcitonin gene-related peptide** (CGRP) for reducing allergen-induced **airway hyperresponsiveness**. **Gelfand, Erwin W.; Dakhama, Azzeddine** (National Jewish Medical and Research Center, USA). PCT Int. Appl. WO 2001068118 A1 20010920, 69 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. English). CODEN: PIXXD2. APPLICATION: WO 2001-US8264 20010314. PRIORITY: US 2000-PV189622 20000314.

AB Disclosed is a method to reduce **airway hyperresponsiveness**, such as allergen-induced **airway hyperresponsiveness**, in a mammal by administering an agent that increases the biol. activity of a CGRP receptor. The agent administered to a mammal is an antibody in conjunction with another agents selected from the group consisting of corticosteroids, (oral, inhaled and injected), .beta.-agonists (long and short acting), leukotriene modifiers (inhibitors or receptor antagonists), antihistamines, phosphodiesterase inhibitors, sodium cromoglycate, nedocromil, theophylline and CGRP. Also disclosed are methods for identifying compds. useful in the present method.

=> s "CGRP"

```
L11      23371 "CGRP"

=> s l11 and lung
L12       1029 L11 AND LUNG

=> s l12 and "AHR"
L13          5 L12 AND "AHR"

=> dup remove l13
PROCESSING COMPLETED FOR L13
L14          2 DUP REMOVE L13 (3 DUPLICATES REMOVED)

=> d l14 1-2 ckib abs

L14  ANSWER 1 OF 2     MEDLINE
2002219908  Document Number: 21952265.    PubMed ID: 11956058.   Regulation of
      airway hyperresponsiveness by calcitonin gene-related peptide in allergen
      sensitized and challenged mice. Dakhama Azzeddine; Kanehiro Arihiko;
      Makela Mika J; Loader Joan E; Larsen Gary L; Gelfand Erwin W. (Division of
      Cell Biology and Pulmonary Medicine, Department of Pediatrics, National
      Jewish Medical and Research Center, Denver, Colorado 80206, USA. )
      AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE, (2002 Apr 15)
      165 (8  1137-44. Journal code: 9421642. ISSN: 1073-449X. Pub. country:
      United States. Language: English.
AB    Sensory neuropeptides are localized to airway nerves and endocrine cells
      in both human and animal species and may participate in the development of
      airway inflammation and hyperresponsiveness (AHR). We used a
      mouse model to identify the changes that occur in calcitonin gene-related
      peptide (CGRP) expression in the airways during development of
      allergic inflammation and to investigate the potential role of this
      neuropeptide in modulating AHR. In sensitized mice, allergen
      challenge induced eosinophilic airway inflammation and AHR and
      resulted in considerable depletion of CGRP in neuroepithelial
      bodies and submucosal nerve plexuses without altering the overall density
      of airway nerve fibers. This depletion was subsequent to the development
      of airway inflammation and was prevented by anti-very late antigen-4 and
      anti-interleukin-5 treatments, which blocked airway eosinophilia and
      abolished AHR. Administration of CGRP to sensitized
      and challenged mice resulted in the normalization of airway responsiveness
      to inhaled methacholine, an effect that was neutralized by the receptor
      antagonist CGRP(8-37). These data demonstrate that replacement
      of CGRP following its depletion in allergic mice can reverse the
      changes in airway responsiveness and suggest that CGRP may have
      potential for the treatment of allergic AHR.

L14  ANSWER 2 OF 2     MEDLINE                                  DUPLICATE 1
2001540883  Document Number: 21471682.    PubMed ID: 11588107.   Chronic
      smoking enhances tachykinin synthesis and airway responsiveness in guinea
      pigs. Kwong K; Wu Z X; Kashon M L; Krajnak K M; Wise P M; Lee L Y.
      (Department of Physiology, University of Kentucky, Lexington, Kentucky
      40536, USA. ) AMERICAN JOURNAL OF RESPIRATORY CELL AND MOLECULAR BIOLOGY,
      (2001 Sep) 25 (3) 299-305. Journal code: 8917225. ISSN: 1044-1549. Pub.
      country: United States. Language: English.
AB    This study tests the hypothesis that the bronchial hyperreactivity induced
      by chronic cigarette smoke (CS) exposure involves the increased expression
      and release of tachykinins and calcitonin gene-related peptide (
      CGRP) from afferent nerve fibers innervating the airways. In
      guinea pigs chronically exposed to CS (30 min twice daily for 14-17 d),
      peak response in total lung resistance to capsaicin (1.68
      microg/kg, intravenously) was significantly greater than that evoked by
      the same dose of capsaicin in control (air-exposed) animals. This
      augmented response in CS-exposed animals was abolished after treatment
      with CP-99994 and SR-48968, the neurokinin (NK)-1 and NK-2 receptor
```

antagonists, suggesting the involvement of tachykinins in chronic CS-induced airway hyperresponsiveness (**AHR**). Further, substance P (SP)-like immunoreactivity (LI) and **CGRP**-LI in the airway tissue were significantly greater in the CS animals than in the control animals. Finally, beta-preprotachykinin (PPT, a splice variant from the PPT A gene encoding tachykinins including SP and NKA) messenger RNA levels as measured by in situ hybridization histochemistry displayed a significant increase in jugular ganglion neurons but not in dorsal root or nodose ganglion neurons. These data suggest that chronic CS-induced **AHR** is related to an increase in SP synthesis and release in jugular ganglion neurons innervating the **lungs** and airways.

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FILE 'MEDLINE, EMBASE, BIOSIS, SCISEARCH, CAPLUS' ENTERED AT 12:56:24 ON 20 JUL 2002

L1 8882 S AIRWAY HYPERRESPONSIVENESS
L2 1246 S L1 AND INHIBITOR
L3 1 S L2 AND CALCITONIN GENE RELATED PEPTIDE RECEPTOR
L4 0 S L2 AND "CGRP" ANTAGONIST
L5 1 S L2 AND "CGRP RECEPTOR"
L6 2914 S (GELFAND E?/AU OR DAKHAMA A?/AU)
L7 265 S L6 AND AIRWAY HYPERRESPONSIVENESS
L8 94 DUP REMOVE L7 (171 DUPLICATES REMOVED)
L9 1 S L8 AND CALCITONIN GENE RELATED PEPTIDE
L10 1 DUP REMOVE L9 (0 DUPLICATES REMOVED)
L11 23371 S "CGRP"
L12 102 S L11 AND LUNG
L13 1 S L12 AND "AHR"
L14 1 DUP REMOVE L13 (0 DUPLICATES REMOVED)

=> s l11 and treatment
L15 2738 L11 AND TREATMENT

=> s l15 and allergy
L16 12 L15 AND ALLERGY

=> dup remove l16
PROCESSING COMPLETED FOR L16
L17 10 DUP REMOVE L16 (2 DUPLICATES REMOVED)

=> d l17 1-10 cbib abs

L17 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2002 ACS
2000:388522 Document No. 133:39703 Nucleic acid encoding a bovine calcitonin receptor-like receptor (BECRLR). Aiyar, Nambi V.; Disa, Jyoti (Smithkline Beecham Corporation, USA). U.S. US 6074845 A 20000613, 16 pp. (English). CODEN: USXXAM. APPLICATION: US 1999-238796 19990128.
AB BECRLR polypeptides and polynucleotides and methods for producing such polypeptides by recombinant techniques are disclosed. Also disclosed are methods for utilizing BECRLR polypeptides and polynucleotides in screening assays to discover compds. that either agonize or antagonize the biol. activity of the receptor. Such compds. are expected to be useful in **treatment** of human diseases, including, but not limited to: infections such as bacterial, fungal, protozoan and viral infections, particularly infections caused by HIV-1 or HIV-2; pain; cancers; diabetes, obesity; anorexia; bulimia; asthma; Parkinson's disease; acute heart failure; hypotension; hypertension; urinary retention; osteoporosis; angina pectoris; myocardial infarction; stroke; ulcers; asthma; **allergies**; benign prostatic hypertrophy; migraine; vomiting;

psychotic and neurol. disorders, including anxiety, schizophrenia, manic depression, depression, delirium, dementia, and severe mental retardation; and dyskinesias, such as Huntington's disease or Gilles de la Tourette syndrome.

L17 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2002 ACS
1999:622267 Document No. 131:253022 Method for counteracting vasospasms, ischemia, renal failure, and treating male impotence using calcitonin gene related peptide. Wimalawansa, Sunil J. (USA). U.S. US 5958877 A 19990928, 9 pp. (English). CODEN: USXXAM. APPLICATION: US 1995-446929 19950518.

AB The present invention provides a method for counteracting pathol. vasospasms or ischemia in target arteries (coronary, carotid, and renal arteries , where the vasospasms or ischemia are due to angioplasty, vascular graft, stent insertion, or arterial surgery by administering calcitonin gene related peptide (**CGRP**) or its analogs. Renal failure and male impotence can also be treated using **CGRP** or its analogs. **CGRP** is a naturally occurring substance in the human body. As such, **CGRP** does not have the same toxicity and **allergy** problems as the foreign substances that currently are used for similar purposes. When locally applied or infused, the effects of **CGRP** are limited to a local vascular area. Virtually no systemic effects are induced, making **CGRP** extremely safe and effective.

L17 ANSWER 3 OF 10 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
1999190541 EMBASE The effect of neuropeptides/hormones on Langerhans cells. Torii H.; Tamaki K.; Granstein R.D.. H. Torii, Department of Dermatology, University of Tokyo, Hongo 7-3-1, Bunkyo-ku, Tokyo 113-8655, Japan. Journal of Dermatological Science 20/1 (21-29) 1999.
Refs: 47.
ISSN: 0923-1811. CODEN: JDSCEI.
Publisher Ident.: S 0923-1811(99)00004-3. Pub. Country: Ireland. Language: English. Summary Language: English.

AB Neuropeptides/hormones have been shown to regulate the various functions of many immunocompetent cells. A number of neuropeptides/hormones has been demonstrated to be present in the skin and a close anatomical association between calcitonin gene-related peptide (**CGRP**)-containing nerves and Langerhans cells (LC) has been reported. In addition to the **CGRP** receptor, receptors for several neuropeptides including pituitary adenylate cyclase activating polypeptide (PACAP) and gastrin releasing peptide (GRP) are found on LC, suggesting these neuropeptides might have some effects on LC. **CGRP** inhibits all antigen presentation and stimulation of a specific-antigen responsive T-cell clone by LC. Pre-**treatment** of LC with **CGRP** also inhibits the elicitation of delayed type hypersensitivity (DTH) in tumor immune mice. Upregulation of B7-2 expression on LC is suppressed by **CGRP**, which might be, in part, responsible for the inhibitory effect of **CGRP** in the functional assay. The production of some inflammatory cytokines such as IL-10 by LC- like cell line XS52 is regulated by **CGRP** and the functional effect of **CGRP** appears to be at least partially mediated through the autocrine regulation of IL-10. .alpha.-MSH is another neuropeptide, the effect of which has been well studied in the cutaneous immune system. Pre-**treatment** of mice with .alpha.-MSH produces inhibitory effects in contact hypersensitivity (CHS). IL-10 has been suggested to be involved in the inhibitory effect of .alpha.-MSH. The receptors and the functional effects of other proopiomelanocortin (POMC)-derived peptides including .beta.-endorphin and catecholamines on LC are under investigation.

L17 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2002 ACS
1999:62193 Document No. 130:131184 Polynucleotides that encode the human calcitonin gene-related peptide receptor component factor (**CGRP**-RCF, HOUND044). Adamou, John E.; Elshourbagy, Nabil (Smithkline Beecham

Corp., USA). U.S. US 5710024 A 19980120, 24 pp. (English). CODEN: USXXAM. APPLICATION: US 1996-686178 19960723.

AB Human **CGRP**-RCP polypeptides and DNA (RNA encoding such **CGRP**-RCP and a procedure for producing such polypeptides by recombinant techniques is disclosed. **CGRP**-RCP is obtained using std. cloning and screening procedures, such as those for cloning cDNAs using mRNA from human adipocytes from osteoblastoma as starting material. Human **CGRP**-RCP cDNA contains an open reading frame encoding a protein of 148 amino acid residues, and is structurally related (83% identity and 94% similarity) to the guinea pig **CGRP**-RCP. Procedures are provided for expression and purifn. of human **CGRP**-RCP using recombinant bacteria, cloning and expression in a baculovirus expression system, and gene therapeutic expression in fibroblasts. Also disclosed are methods for utilizing such **CGRP**-RCP for the **treatment** of diabetes, migraine, pain and inflammation, Parkinson's disease, acute heart failure, hypotension, urinary retention, osteoporosis, hypertension, angina pectoris, myocardial infarction, ulcers, asthma, **allergies**, psychosis, depression, vomiting, benign prostatic hypertrophy, Paget's disease, obesity, cancer, gigantism and the like. Antagonists against such **CGRP**-RCP and their use as a therapeutic to treat diabetes, migraine, pain and inflammation, Parkinson's disease, acute heart failure, hypotension, urinary retention, osteoporosis, hypertension, angina pectoris, myocardial infarction, ulcers, asthma, **allergies**, psychosis, depression, vomiting, benign prostatic hypertrophy, Paget's disease, obesity, cancer, gigantism and the like are also disclosed. Also disclosed are diagnostic assays for detecting diseases related to mutations in the nucleic acid sequences and altered concns. of the polypeptides. Also disclosed are diagnostic assays for detecting mutations in the polynucleotides encoding the **CGRP**-RCP and for detecting altered levels of the polypeptide in a host.

L17 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2002 ACS

1997:643681 Document No. 127:343635 **Treatment** of preeclampsia, eclampsia or preterm labor with calcitonin gene related peptide and related drugs. Yallampalli, Chandrasekhar; Wimalawansa, Sunil J. (Board of Regents, the University of Texas System, USA; Yallampalli, Chandrasekhar; Wimalawansa, Sunil J.). PCT Int. Appl. WO 9734922 A1 19970925, 87 pp. DESIGNATED STATES: W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1997-US4310 19970318. PRIORITY: US 1996-619841 19960318.

AB The present invention provides a method for counteracting preeclampsia, eclampsia of pregnancy and preterm labor in a pregnant female mammal treated by administering thereto calcitonin gene-related peptide (**CGRP**) or its analogs including **CGRP**/adrenomedullin or their peptide or receptor-based analogs, or in combination with a progestin, and with or without a nitric oxide substrate, or a nitric oxide donor or both, optionally in further combination with one or more of a cyclooxygenase inhibitor, a PGI2-mimetic, a thromboxane (TXA2) inhibitor, a compd. possessing TXA2-agonistic and TXA2-inhibiting properties, a compd. possessing TXA2-antagonistic and PGI2-mimetic activities, and a TXA2 antagonist. **CGRP**, progesterone and nitric oxide substrate and/or any nitric oxide donor compds., alone or in combination can be used. As **CGRP** is a naturally occurring compd., it does not have the same toxicity and **allergy** problems as the foreign substances that are currently used for similar purposes. During pregnancy uterine blood vessels and the uterine muscles are extremely sensitive to **CGRP** as well as nitric oxide. Therefore, one could administer a very small quantities of these drugs i.e., i.v., s.c., or implants), the

effects are then seen mainly in the uterine muscle and blood vessels, namely increase the blood supply to the utero-placental unit (hence nutrients and oxygen supply to the fetus through the improved placental circulation), and uterine muscular relaxation thereby ameliorate the signs and symptoms of preeclampsia, and eclampsia, and prevent preterm labor. At these dosages, virtually no systemic effects are induced, making **CGRP** (which is an endogenous natural product present in human body) extremely safe and effective.

L17 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2002 ACS
1997:346284 Document No. 126:334212 Cosmetic and pharmaceutical compositions containing salts of lanthanide, tin, zinc, manganese, yttrium, cobalt, strontium as substance P antagonists. Breton, Lionel; De Lacharriere, Olivier (Oreal S. A., Fr.). Eur. Pat. Appl. EP 770393 A2 19970502, 10 pp. DESIGNATED STATES: R: AT, BE, CH, DE, ES, FR, GB, IE, IT, LI, NL, SE. (French). CODEN: EPXXDW. APPLICATION: EP 1996-402183 19961014. PRIORITY: FR 1995-12658 19951026.

AB The title cosmetic and pharmaceutical compns. are claimed for **treatment** of disorders assocd. with excess synthesis or release of substance P. A lotion contained manganese chloride 15.00, glycerol 2.00, Me paraben 0.15, perfume q.s., and water q.s. 100.00.

L17 ANSWER 7 OF 10 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
97231534 EMBASE Document No.: 1997231534. Calcitonin gene-related peptide and Langerhans cell function. Torii H.; Hosoi J.; Asahina A.; Granstein R.D.. Dr. R.D. Granstein, Department of Dermatology, Cornell University Medical College, 525 East 68th Street, New York, NY 10021, United States. Journal of Investigative Dermatology Symposium Proceedings 2/1 (82-86) 1997. Refs: 37.
ISSN: 1087-0024. CODEN: JDSPPO. Pub. Country: United States. Language: English. Summary Language: English.

AB Morphologic studies have indicated that Langerhans cells (LC) are frequently in anatomic apposition with epidermal nerves containing the neuropeptide calcitonin gene-related peptide (**CGRP**). Experiments were undertaken to examine whether **CGRP** modulates LC function. The effect of pre- exposure of LC to **CGRP** in vitro on allogeneic antigen presentation and specific protein presentation to a responsive T-cell line were studied using freshly prepared murine epidermal cell populations enriched for LC content (EC) by **treatment** with antibody to Thy-1 and complement. The ability of EC to present tumor-associated antigens for induction and elicitation of delayed-type hypersensitivity (DTH) in tumor-immune mice was also examined. Inhibitory effects of **CGRP** on antigen presentation were observed in each of these assays. Experiments were also performed examining the ability of intradermally administered **CGRP** to modulate induction of contact hypersensitivity (CHS) to a hapten applied at the injected site. Administration of **CGRP** led to a decrease in the CHS response after immunization at the site of injection. Intracellular cAMP was significantly increased in freshly prepared LC after exposure to **CGRP**, and this process could be inhibited by a specific inhibitor of the **CGRP** receptor, demonstating the existence of **CGRP** receptors on LC. B7-2 expression induced by LPS and GM- CSF in the LC-like line XS52, and by LPS in peritoneal macrophages, was suppressed by **CGRP**. This suppression may account, in part, for the inhibitory effect of **CGRP**. As a whole, these observations suggest that regulation of antigen presentation by nerves may occur in the epidermis.

L17 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2002 ACS
1996:666998 Document No. 125:284978 Use of a calcitonin gene-related peptide antagonists for the **treatment** of ocular or eyelid pruritus and dysesthesia. De Lacharriere, Olivier; Breton, Lionel (Oreal S. A., Fr.). Eur. Pat. Appl. EP 734730 A1 19961002, 9 pp. DESIGNATED STATES: R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE. (French).

CODEN: EPXXDW. APPLICATION: EP 1996-400459 19960304. PRIORITY: FR 1995-3629 19950328.

AB Pharmaceutical or cosmetic compns. contg. calcitonin gene-related peptide (**CGRP**) antagonists are used for the **treatment** of ocular or eyelid pruritus and dysesthesia. A collyrium contained **CGRP** 8-37 0.5, and excipients comprising sodium chloride, sodium borate, Polysorbate 80, boric acid and water q.s. 100.

L17 ANSWER 9 OF 10 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

91101225 EMBASE Document No.: 1991101225. Airways vasodilatation in the immediate allergic reaction. Involvement of inflammatory mediators and sensory nerves. Alving K., Department of Pharmacology, Karolinska Institutet, S-104 01 Stockholm, Sweden. Acta Physiologica Scandinavica, Supplement 141/597 (1-64) 1991.
ISSN: 0302-3994. CODEN: ABSSAD. Pub. Country: United Kingdom. Language: English. Summary Language: English.

AB 1. Systemic capsaicin **treatment** of the pig depletes the content of sensory neuropeptides (**CGRP** and tachykinins) in the airways mucosa and skin, without affecting sympathetic and parasympathetic nerves containing NPY and VIP, or the presence and appearance of inflammatory cells including mast cells. Acute capsaicin exposure caused release of sensory neuropeptides and catecholamines, and marked vasodilatation in the airways and skin, without signs of plasma protein extravasation or bronchoconstriction. Capsaicin pretreatment effectively desensitizes against local challenges with capsaicin in the airways and skin, as revealed by the absence of vasodilatory responses 2 days later. 2. Cigarette smoke exposure induces marked vasodilatation, lasting for about 5 min in both the upper and lower airways, which seems not to be primarily caused by particulate matter or nicotine in the smoke. Except for a minor capsaicin-sensitive component in the nasal circulation, these responses probably do not involve neural activation, mast cell degranulation or prostaglandin formation. Rather, it is concluded that vapour phase components act on the vessels via unknown mechanisms. 3. Sensitization of pigs with s.c. injections of ascaris antigen was successful, resulting in typical wheal and flare reactions in the skin and bronchoconstriction after local challenge with antigen. The reactivity to ascaris is probably mediated by antibodies of the IgE isotype. 4. Histamine-containing mast cells and sensory neuropeptide-containing nerve fibres show close morphological association around blood vessels in the pig skin. Both alcian blue-positive mast cells and capsaicin-sensitive sensory nerves are present close to the pig airways epithelium. Sensory neuropeptide-containing nerves are also abundant around airways mucosal blood vessels, whereas the bronchial smooth muscle is sparsely innervated. 5. Allergen and histamine injections in the skin caused similar responses consisting of flare and protein extravasation. Allergen challenge in the airways induces marked vasodilatation lasting for 60-90 min in the pig bronchial and nasal circulations. Histamine seems to be important in the early phase (0-20 min) of these responses in the airways, while cyclo-oxygenase products (possibly PGD2) may be responsible for the long-lasting component. A cyclo-oxygenase product is presumably also released from the lung into the circulation after bronchial allergen challenge and thereby induces a delayed, long-lasting nasal vasodilatation. Histamine may be the main bronchoconstrictor agent released in the immediate allergic reaction of the pig. 6. The flare, but not the protein extravasation reaction, to allergen and histamine injections in the skin, was inhibited by capsaicin pretreatment. Aerosolized histamine and bradykinin, but not PGD2, seem to cause airways vasodilatation partly via activation of capsaicin-sensitive sensory nerves. Nedocromil sodium reduces the airways vasodilatation response to bradykinin and the histamine-induced nasal vasodilatation in the same manner as capsaicin pretreatment. It may thus be suggested that nedocromil sodium acts at least partly by inhibiting sensory nerve activation by inflammatory mediators. Nedocromil sodium also reduced the allergen-induced bronchoconstriction, indicating an inhibitory action on

mast cells as well. Capsaicin-sensitive sensory nerves are probably mainly involved in the airways vasodilatation seen after allergen challenge. 7. In conclusion, a large animal model has been developed where the influence of irritants and allergen on arterial blood flow at three different levels of the airways can be monitored in parallel to pulmonary mechanics. Furthermore, by using systemic capsaicin pretreatment, the involvement of capsaicin-sensitive sensory nerves in airways reactions can be studied. The pig seems to be more related to man, regarding sensory nerve-mediated protein extravasation and bronchoconstriction, compared to the rat and guinea pig. The airways vasodilatation in the immediate allergic reaction may primarily involve histamine and PGD2. Part of the response is probably mediated via activation of capsaicin-sensitive sensory nerves by mediators such as histamine and bradykinin. Cigarette smoke-induced vasodilatation may be caused by vapour phase components acting on the vessels.

L17 ANSWER 10 OF 10 MEDLINE DUPLICATE 1
87182279 Document Number: 87182279. PubMed ID: 3494409.
Capsaicin-sensitive nerves and the cutaneous **allergy** reaction in man. Possible involvement of sensory neuropeptides in the flare reaction.
Lundblad L; Lundberg J M; Anggard A; Zetterstrom O. ALLERGY, (1987 Jan) 42 (1) 20-5. Journal code: 7804028. ISSN: 0105-4538. Pub. country: Denmark. Language: English.

AB The effects of local capsaicin pretreatment on the cutaneous triple response reaction induced by allergen exposure or anti-IgE were studied in man. Acute exposure of the human skin to capsaicin caused a burning sensation and a clearcut flare reaction but no wheal response. Upon repeated administration these local reactions to capsaicin disappeared. The flare component and the subjective itching sensation of the cutaneous **allergy** reaction to rat antigen in sensitized persons or anti-IgE in non-allergic persons were then markedly reduced. Two weeks after capsaicin pretreatment the flare response to allergen was not significantly changed compared to the control reaction, suggesting a reversible effect of capsaicin **treatment**. The wheal component of the **allergy** or anti-IgE reaction was, however, not influenced by capsaicin pretreatment, indicating that the wheal and flare components are caused by different mechanisms. It is concluded that capsaicin sensitive sensory nerves are of importance for the human cutaneous triple response reaction induced by allergen exposure. Thus, secondary release of mediators, such as **CGRP** or tachykinins from sensory nerve branches, may contribute to the flare component of this reaction. Furthermore the itching sensation seems to be dependent to a large extent on capsaicin-sensitive nerves. However, sensory nerves seem to have less importance for the wheal reaction, i.e. the protein extravasation response.

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Executing the logoff script...

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	77.76	77.97

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-5.58	-5.58

SESSION WILL BE HELD FOR 60 MINUTES

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